More Lack in the World

The Complex Connection between Undernutrition and Climate Change

Anthropogenic climate change is projected to reduce cereal yields and food security and therefore to undermine future efforts to reduce child undernutrition. But models are needed to better measure the potential impacts of climate change on population health. Now researchers have developed a model to estimate future undernutrition attributable to climate change as a function of its impact on crop productivity [***EHP* 119(12):1817–1823; Lloyd et al.**].

Undernutrition is measured using criteria such as stunting (smaller-than-average height-for-age) and underweight (smaller-than-average weight-for-age). The researchers developed and validated the model using previously published data about past food availability, the prevalence of stunting, and gross domestic product. Then they used projections of future calorie availability under two climate change scenarios and a reference scenario of no climate change to estimate undernutrition among children under age 5 years in five regions of South Asia and sub-Saharan Africa in 2050.

The model estimates that climate change will lead to an average relative increase in moderate stunting (height that is more than two standard deviations below the expected height-for-age) of 1–29%, depending on region, compared with a future without climate change. Climate change will have a greater impact on rates of severe stunting (height more than three standard deviations below the expected mean), which the authors estimate will increase by an average of 31–62%, depending on region.

A Ugandan child scrapes out extra food from a cooking pot. Climate change is expected to undermine efforts against child undernutrition by reducing cereal yields and food security.

Climate change is likely to affect undernutrition through a variety of means in addition to crop production, including impacts on infectious diseases in humans, plant pests and diseases, labor productivity, and water availability. One limitation of the current study is the difficulty in quantifying the impact of climate change in the face of uncertainty about how countries will develop and manage their food systems. The authors state that their current study illustrates the importance of the outcome used to predict impacts—undernourishment (lack of food) versus stunting, for instance, or moderate versus severe stunting have different implications for decision making and for population health.

The study adds to the evidence suggesting that climate change is likely to increase future hunger and undernutrition even under optimistic assumptions of future emissions and economic growth. The study results suggest that to reduce and prevent future undernutrition, it is necessary to not only reduce emissions of greenhouse gases but also increase food access and improve socioeconomic conditions.

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Hormone Impact

BPA Linked to Altered Gene Expression in Humans

Urinary metabolites of bisphenol A (BPA), a widely used component of polycarbonate plastics and epoxy resins, serve as biomarkers for exposure to the chemical and are detectable in more than 90% of individuals tested in the United States and Europe. Studies to date suggest positive associations between BPA and cardiovascular disease, diabetes, and reproductive and developmental abnormalities in humans, although further research is needed to confirm these findings. Recent studies have shown links between BPA and changes in total testosterone concentrations and altered estradiol:testosterone ratio in men, but evidence for a mechanism behind such links has been lacking. A new study now links BPA exposure with altered expression of estrogen- and androgen-responsive genes in humans [***EHP* 119(12):1788–1793; Melzer et al.**].

The InCHIANTI study—a prospective study of mid- and late-life morbidity risk factors among 1,453 participants in Chianti, Italy—provided the data for the current study. A subset of 96 men provided same-day blood and urine samples in 2008–2009. Urine samples were analyzed for concentrations of BPA, and blood leukocytes were used for transcript analysis of six estrogen- and androgen-responsive genes: *ESR1*, *ESR2*, *ESRRA*, *ESRRB*, *ESRRG*, and *AR*. These genes code nuclear hormone receptors involved in the control of developmental and physiological pathways shown to be activated by BPA in laboratory studies.

Urinary BPA concentrations ranged from 0.73 to 56.94 ng/mL and were positively associated with increased expression of *ESR2* and *ESRRA* based on models adjusted for potential confounding factors. Transcripts for other genes were either not detected (*ESRRG*) or were not associated with BPA concentrations (*ESR1*, *ESRRB*, and *AR*). Mean expression of *ESR2* and *ESRRA* increased by 65% and 38%, respectively, in the highest versus lowest BPA exposure tertile.

The implications of altered gene expression in blood leukocytes are unknown, and this measure has not been validated as a surrogate measure of effects on hormone-responsive gene expression. However, the results suggest that BPA is bioactive in humans, and the authors argue that the potential link between exposure, hormone signaling, and related disorders is biologically plausible. For example, estrogen receptor β, coded by *ESR2*, plays a significant role in maintaining the structure and function of tissues in the cardiovascular and central nervous systems.

The cross-sectional design, lack of distinction between free and conjugated BPA in urine samples, and possible unidentified confounding factors are limitations of the study. Additional research is needed to confirm the findings and further investigate gene expression changes and effects of BPA exposure in other estrogen-regulated target tissues.

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Artificial Food Color Additives and Child Behavior: Weiss Responds

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The Food and Drug Administration's (FDA) response to my commentary (Weiss 2012) reflects the wide gulf between how the FDA translates "weight of evidence" into regulatory policy for artificial food colors (AFCs) and how it is translated into meaningful action on behalf of public protection.

The FDA essentially took the position that for a study to be considered as evidence of adverse effects, it must be totally free of uncertainties. The study by McCann et al. (2007) played a large role is provoking the FDA review, but for that study, like almost any epidemiological study, it would be difficult to meet that absolute criterion. It is why *Environmental Health Perspectives (EHP)* publishes so many such studies addressing the same question (e.g., air pollution). But isn't it fair to ask whether any of the negative AFC studies meet that criterion?

In their critique, the FDA faults McCann et al. (2007) because they characterized "… a treatment effect as adverse when it may, in fact, fall within the normal range of childhood behavior." This is an issue discussed over and over again in the pages of *EHP*. Take the example in my commentary (Weiss 2012), modeled on numerous publications in the lead literature (e.g., Lanphear et al. 2005): If developmental exposure to low levels of lead reduces a population IQ (intelligence quotient) by 3 points (3%), from, say, 100 to 97, it is taken as evidence of a major adverse effect. Both scores, of course, fall within the normal range. The same criticism is used by the FDA to dismiss the effect size calculations; that is, the altered behavioral activity seen in published data lies "… in the range of normal activity for children."

The FDA finds the study by McCann et al. (2007) lacking because the authors relied mainly on parental observations. A high proportion of child development research, in fact, enlists parents as observers; hundreds of validated inventories and questionnaires are based on parent ratings. They are the observers, of course, who see the most extensive samples of the child's behavior, especially with younger children. This is the reason I chose parental observations for my own food color study of young children (Weiss et al. 1980) and why we relied on parent ratings for our study of how phthalates mold play behavior in preschool children (Swan et al. 2010).

It is difficult to grasp the FDA argument that AFCs do not possess "inherent" neurotoxic properties but may provoke neurotoxicity in susceptible subpopulations. Neurotoxicity is neurotoxicity.

The FDA does acknowledge that AFCs may be associated with adverse behavioral outcomes in some (unknown proportion of) susceptible children. As I note in my commentary (Weiss 2012), such a conclusion would prompt decisive action by the U.S. Environmental Protection Agency. Why not the FDA?

I was pleased to hear that the FDA noted the need for further research. My question remains: What parent or institutional review board (IRB) would be convinced that such research is without significant risk, given what we already know? If IRBs would hesitate, shouldn't that prompt the FDA to at least require warning labels on foods containing AFCs that are consumed mainly by children?

Finally, the FDA policy reflects a point of view that is endemic in federal regulatory policy toward potentially toxic chemicals. Namely, a chemical is innocent until proven guilty. Many environmental health researchers believe the proposition needs to be reversed. Some advocate adoption of the precautionary principle. Perhaps, if the FDA had required neurotoxicity testing, especially in young children, before allowing AFCs and other additives to be marketed, we would not be having this debate at all. Harvey Wiley, who became the FDA's first commissioner, recruited his legendary "Poison Squad" volunteers for precisely this purpose. That was in 1902.

The author declares he has no actual or potential competing financial interests.

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Erratum

The December Science Selections articles "More Lack in the World" [Environ Health Perspect 119:A524 (2011); http://dx.doi.org/10.1289/ehp.119-a524a] and "Full of Beans?" [Environ Health Perspect 119:A525 (2011); http://dx.doi.org/10.1289/ehp.119-a525b] mistakenly reversed the page numbers for the associated research articles. The December Forum article "NY DEC Takes on Fracking" [Environ Health Perspect 119:A513 (2011); http://dx.doi.org/10.1289/ehp.119-a513] incorrectly suggested that the public comment period for the New York Department of Environmental Conservation's Supplemental Generic Environmental Impact Statement had already closed. *EHP* regrets the errors.